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Critical Dynamics in Homeostatic Memory Networks

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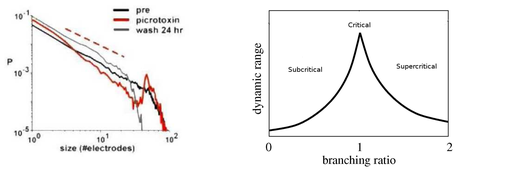
Criticality in the Brain

The brain can be described best as a constantly shifting complex dynamic system with an inherent mechanism favouring **self-organisation**, where in a large repertoire of spatiotemporal acivity patterns in the brain is the basis for adaptive behaviour.

It has also been proposed that the brain self-organises for criticality i.e. the near critical behaviour is the consequence of the synaptic plasticity rules involved in learning [1].



picture taken and adapted from [4]

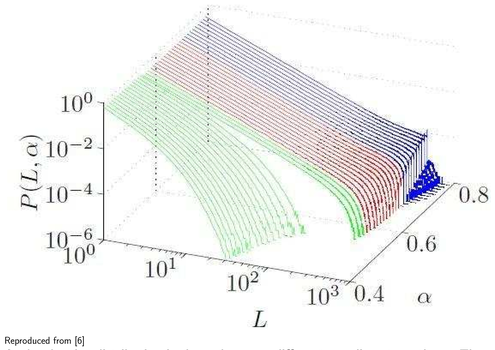
- Spontaneous Brain dynamics is complex
- The complexity of brain dynamics is a signature of the underlying critical process Chialvo2001
- Experimental evidence of neuronal populations exhibiting critical dynamics : cortical quakes
- Higher brain functions such as perception, learning and goal directed movement are often hypothesized to depend on the collective dynamics of large number of interacting neurons distributed throughout the cortex

Neuronal Avalanches

Using a model of integrate and fire neurons with activity dependent synapses it can be shown that several dynamic regimes exist in reconciliation with parameter independent criticality. Synaptic depression causes the mean synaptic strength to approach a critical value for a certain range of values of interaction parameters where in other dynamical behaviours are prevalent outside this range.

The critical state is characterised by a **power law statistics of neural avalanche sizes**.

Depressive synapses cause a broad and stable critical regime in integrate-and-fire networks independent of parameter settings [6].



Reproduced from [6]

Avalanche size distribution in dependence on different coupling strengths α. The critical regime is shown in red.

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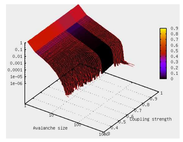
Abstract

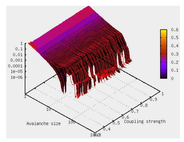
Critical behavior in neural networks characterized by scale-free event distributions and brought about by self- regulatory mechanisms such as short-term synaptic dynamics or homeostatic plasticity, is believed to optimize sensitivity to input and information transfer in the system. Although theoretical predictions of the spike distributions have been conrmed by in-vitro experiments, in-vivo data yield a more complex picture which might be due to the in-homogeneity of the network structure, leakage in currents or massive driving inputs which has so far not been comprehensively covered by analytical or numerical studies. We address these questions by the study of a neural model of memory that allows for storage and retrieval of patterns and for recombining such patterns as needed for search in problem solving. The model features critical dynamics in the neural assembly as a result of the interplay of synaptic depression and facilitation (Levina e.a 2007, 2009). Model simulations show that the prolonged consolidation of memory patterns induces a bias towards the memories which affects the scale- free spike-frequency distribution. However Selective modication of neuronal circuitry in the form of controlled homeostasis of synaptic weights is seen to mitigate this bias such that the network self-organizes to maintain the average strength of the synapses at the critical level. The resulting spike statistics depends on the assumed coding scheme, but even sparse or orthogonal memory patterns introduce a typical event size which is incompatible with critical dynamics below the maximal memory capacity. The combination of memory and ongoing dynamics in the model was chosen for its implications in the context of cognitive aging. Following the paradigm of aging as a multi-criteria optimization process, we posit aging effects as a result of an increasing incompatibility of learning goals.

Results

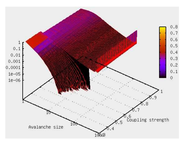
Here we show exemplary results of our simulations for different pattern sets. Parameters were in all four cases: $N = 300,\ 500$, $\tau_J = 0.02$, $u_0 = 0.1$, and the value of α varies from 0.33 to 0.99 incremented in steps of 0.01



Single Pattern

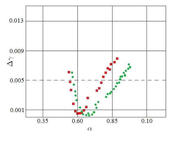


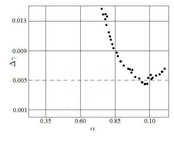
Random Multiple Patterns

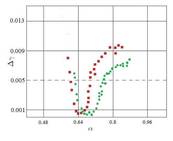


De-correlated patterns

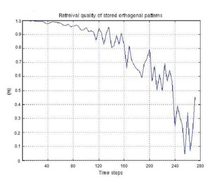
Note the varying avalanche distributions (log-log scale) with varying α values, for the three cases. The single distinct pattern case shows a clear power law slope with an interesting region in the middle of the critical regime (black) showing an absence of activity. The dynamics with multiple patterns show a small section corresponding to the subcritical regime followed by a sudden jump into a highly structured formation (synchronous wave like formations). Interestingly the decorrelated patterns demonstrate an immediate jump from subcritical regime to a state of supercriticality.

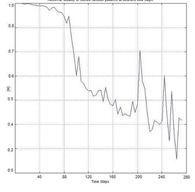






The mean squared deviation from the best matching power law is plotted in dependence with α. 0.005 is set as the threshold indicating power law.The squares and circles stand for networks with depressive and facilitative synapses and system sizes $N = 300,\ 500$ respectively.





Change in stored pattern retrieval quality(m) dynamics (overlaps of the memory states) at different time steps during the learning procedure is plotted.The V-shaped curve in the figure (Right) should correspond to an attractor state denoting high degree of overlap between the stored patterns. Resulting in switching between patterns with an eventual rise in retrieval quality. Over time this behaviour continues – directly relating with the observed loss is criticality.

Neural Model

The membrane potential h_i of neuron i gets discrete external input and receives action potentials from other neurons scaled with the strength of the particular synapse.

$$\dot{h}_i = \delta_{i,\xi_i(t)} I^{\text{ext}} + \sum_{j=1}^{N} J_j \cdot w_{i,j} \delta(t - t^j_{sp} - \tau_d)$$

Synapses are subject to loss of neurotransmitter upon transmission of a spike. The efficacy J is reduced by a fraction u_J while, in the absence of spikes, the synapse recovers to the maximum strength W_{ij} with a slow time rate τ_J.

We use a Hebbian correlation rule for memorisation of the input patterns.

$$W_{ij} = \frac{1}{Nf(1-f)} \sum_{\mu=1}^{P} (\xi^\mu_i - f)(\xi^\mu_j - f); f = \frac{\sum \xi^\mu_i}{N}$$

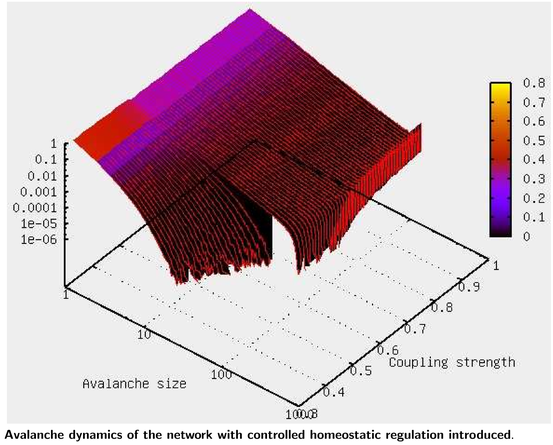
$$\dot{J}_{ji} = \frac{1}{\tau_J}(W_{ij} - J_{ji}) - u_J \cdot J_{ji} \cdot \delta(t - t^j_{sp})$$

Homeostatic regulation is used to control the average synaptic strength obtained from Hebbian learning which partially removes the negative effects of learning on criticality by the following self-regulatory mechanism.

$$\dot{W}_{ji} = \dot{W}_{ij} = \sum_{j=1}^{N} k_\alpha(\alpha - W_{ij})\delta(t - t^j_{sp})$$

Homeostatic Regulation

- **Though Hebbian mechanisms are crucial for the selective modification of neuronal circuitry, they may not be sufficient.**
- **It was noted that chronologically reducing inhibition in cortical networks initially raises firing rates, but they eventually stabilise to control levels.** [9]
- **Interestingly, in a critical network the concept of mean firing rate becomes obscure. The dynamics of the system is characterised by irregular firing of neurons and as a result the mean firing rate is not a reliable characterisation of the neural activity.**



Avalanche dynamics of the network with controlled homeostatic regulation introduced.

Here, after the occurrence of a spike as the amount of neuro-transmitter approaches the maximum synaptic strength, the synaptic weights $W_{i,j}$ between two neurons is regulated to move towards the critical value α. Hence there is a counter balancing of the Hebbian plasticity by this type of synaptic adaptation beyond standard Hebbian learning.

Where, $\alpha^{t+1}_{i,j} = \alpha^t_{i,j} + \epsilon(1 - l)$ is the learning rule used for the critical parameter.

Evaluating the Network

Learning

- A number of patterns (subsets of neurons) are chosen.
- The same set of patterns is shown the entire learning procedure.
- The simulations are carried out in three steps covering the three models: Network with dynamic synapses without associative recall, dynamic synapses with associative recall for orthogonal and random patterns, model with homeostatic regulation.

Testing

After 100 presentations of all patterns the current state of the network is tested for two primary properties:

- **Mean squared deviation from power law:** The loss of criticality is measured by fitting error to a power law.

$$\delta\gamma = \frac{1}{N/2} \sum_{l=2}^{N/2} [lnP(l) - a(lnl + b)]^2$$

- **Quality of retrieval of stored patterns:** The degree of overlap of the stored patterns in conjunction with the mean squared deviation from power law statictics.
- The closeness to a certain pattern ϵ^μ is characterised by an overlap

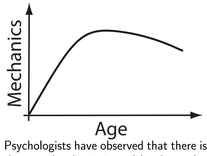
$$m^\mu = \frac{1}{Nf(1-f)} \sum_{i=1}^{N} \eta_i s_i(t); \eta_i = \xi^\mu_j - f$$

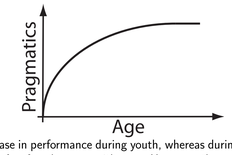
Cognitive Aging: Effect of longer learning history

Several parameters of neural models have been analysed in order to account for ageing in correspondence to known physiological changes in the brain:

- Reduction in the number of neurons
- Reduction of white matter → number of synapses [5]
- Suboptimal neuromodulation (dopamine)
- Reduction of cognitive control
- Increase in noise (stochastic resonance) [7]
- Longer learning history [8]

Here we primarily focus on the effect of a longer learning history on the network dynamics. The prolonged effect of external inputs is seen to interfere with the self-organised critical behaviour of neural activity [2, 3, 6]. While critical behaviour is known to optimise capacity, flexibility and sensitivity of brain function, it is opposed by the stabilisation of memory structures that result from the formation of associative memory such that ageing appears as a conflict between different optimisation principles.





Psychologists have observed that there is a global increase in performance during youth, whereas during ageing, tasks that can be discriminated by demanding skills or **mechanics** decrease with age. However, those tasks requiring **Pragmatics** don't decrease but gradually saturate with age.

Conclusion and Outlook

- We have studied a combination of Hebbian learning with the phenomenon of self-organised criticality.
- Prolonged learning tends to destroys the criticality which may account for the reduction of fluid intelligence with age
- We give an indication of adaptation of neural networks towards a connectivity scheme that helps to maintain criticality.

Although the network considered are idealised, we explored the concept of realistic leaky neurons by introducing homeostatic regulation in the form of synaptic adaptation of weights in addition to Hebbian learning. We advocate that similar homeostatic mechanisms could be responsible for stabilising neuronal circuitry, ensuring criticality and thus preventing the predominance of supercritical regimes as a result of prolonged learning. There seems to be an optimisation process in the brain where in the connectivity scheme evolves in a way to modulate criticality with an eventual trade off between different competing features like working memory, speed of processing, selective retention.

Future research will be directed towards understanding the deterioration of memory retrieval in the context of cognitive aging, having introduced leakagage in the network. Moreover ongoing work is being carried out to test the effect gradual of loss of memory or random noisy patterns on the critical dynamics of the network.

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